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=> s monocyte and multipotent cell
L1 46 MONOCYTE AND MULTIPOTENT CELL

=> s l1 and cd14
L2 2 L1 AND CD14

=> s l2 and cd34
L3 1 L2 AND CD34

=> s l3 and cd45
L4 0 L3 AND CD45

=> s l1 and cd45
L5 1 L1 AND CD45

=> s l1 and collagen type I
L6 0 L1 AND COLLAGEN TYPE I

=> s l2
L7 2 L2

=> s l2 and collagen
L8 0 L2 AND COLLAGEN

=> s monocyte and collagen j
L9 0 MONOCYTE AND COLLAGEN J

=> s l1 and fibronectin
L10 0 L1 AND FIBRONECTIN

=> s l1 and osteoblast
L11 1 L1 AND OSTEOBLAST

=> disp l11 ibib abs 1-1

L11 ANSWER 1 OF 1 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on
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ACCESSION NUMBER: 2000:394976 SCISEARCH

THE GENUINE ARTICLE: 316VG

TITLE: On the track of a human circulating mesenchymal stem cell
of neural crest origin

AUTHOR: Labat M L (Reprint); Milhaud G; Pouchelet M; Boireau P

CORPORATE SOURCE: Ecole Natl Vet, INRA, AFSSA, INRA, UMR 956, 7 Ave Gen
Gaulle, F-94704 Maisons Alfort, France (Reprint); Ecole
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Alfort, France; CHU St Antoine, Dept Biophys, F-75012

Paris, France; INSERM, Serv Audiovisuel, F-78116 Le
Vesinet, France
COUNTRY OF AUTHOR: France
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PUBLISHER: EDITIONS SCIENTIFIQUES MEDICALES ELSEVIER, 23 RUE LINOIS,
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ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The neural markers present in the normal circulating moncytoid cells able, in pathological situations, to transdifferentiate into different mesenchymal-type cells, confirm the hypothesis previously raised that these cells derive from the neural crest. In culture, the normal cells display a great plasticity very reminiscent of microglial cells in culture. Almost a quiescent cell in normal individuals, this moncytoid cell shows its division potentialities in pathological situations of fibrosis and cancer (chondrosarcoma) where it is found to spontaneously proliferate. While the normal neofibroblasts are rapidly recognized and destroyed by fibrophagic T-lymphocytes, the pathological cells escape this control and, as a result, they accumulate in vitro giving rise to a tissue sometimes organized as nodules. Although basically the transdifferentiation process is similar in all the pathological situations of fibrosis and cancer studied so far, the end-result phenotype evokes the pathology the patient is suffering from. It evokes ***osteoblasts*** in a case of osteomyelosclerosis, chondrocytes in a case of chondrosarcoma, myelofibroblasts in a case of fibrosis of lung and kidney in a patient under cyclosporine treatment. Hence, this circulating moncytoid cell is a ***multipotent*** ***cell*** with great division potentiality. These are characteristics of stem/preprogenitor cells. Since this circulating moncytoid cell also bears the neural markers we called it a moncytoid ectomesenchymal stem/preprogenitor cell. Therefore, the existence of an ectomesenchymal system is discussed here. The circulating moncytoid ectomesenchymal stem/preprogenitor cell might be involved in the normal cicatrization process while the fibrophagic T lymphocytes might be involved in its termination. Impairment of this controlled mechanism might result in the development of fibrosis and/or cancer such as chondrosarcoma in vivo. Interestingly, at least in vitro, proliferation is restricted to the moncytoid cell before transdifferentiation takes place. In this model, fibrosis and cancer might share some common steps going from the proliferation of the moncytoid cells to their transdifferentiation into mesenchymal-type cells and the accumulation of these transdifferentiated cells in the tissues. Then, cancer might be distinguished from fibrosis by the additional acquisition of the ability to proliferate by the transdifferentiated cells. The moncytoid ectomesenchymal stem/preprogenitor cell might also be involved in brain neurodegenerative diseases characterized by an accumulation of microglia. The circulating moncytoid ectomesenchymal stem/preprogenitor cell appears as a target for gene therapy in pathological situations of fibrosis and/or cancer where it proliferates out of control. If the normal cell can be expanded and if its transdifferentiation can be directed, the circulating moncytoid ectomesenchymal stem/preprogenitor cell may become a useful tool for cellular therapy, in case of failure in wound healing and tissue regeneration. (C) 2000 Editions scientifiques et medicales Elsevier SAS.

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COPYRIGHT (C) 2007 Elsevier Science B.V., Amsterdam. All rights reserved.=> s 11 and adipocyte
L12 0 L1 AND ADIPOCYTE=> s monocyte and multipotent cell
L13 46 MONOCYTE AND MULTIPOTENT CELL=> s 113 and adipocyte
L14 0 L13 AND ADIPOCYTE=> s 113 and myoblast
L15 1 L13 AND MYOBLAST=> s 113 and chondrocyte
L16 0 L13 AND CHONDROCYTE=> s 113 and myocardia
L17 0 L13 AND MYOCARDIA

=> disp l15 ibib abs 1-1

L15 ANSWER 1 OF 1 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on
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THE GENUINE ARTICLE: 033PH

TITLE: ***Multipotent*** ***cells*** of monocytic origin
improve damaged heart functionAUTHOR: Dresske B (Reprint); El Mokhtari N E; Ungefroren H; Ruhnke
M; Plate V; Janssen D; Siebert R; Reinecke A; Simon R;
Fandrich FCORPORATE SOURCE: Univ Schleswig Holstein, Dept Gen & Thorac Surg, Campus
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ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Recently, we generated cells with multipotent properties from blood ***monocytes*** that in vitro differentiate into various somatic cell types. This experimental study investigated whether these programmable cells of monocytic origin (PCMO) succeed to restore left ventricular function after myocardial infarction (MI). PCMO were generated from ***monocytes*** by exposition to RPMI medium containing M-CSF and IL-3 for 6 days. MI was induced in female Lewis rats ligating the left coronary artery. PCMO of male Lewis donors were injected either intramyocardially (i.my.) or intravenously (i.v.) 24 h or 6 days post-infarction. Hemodynamic assessment after 60 days demonstrated significant improvement of left ventricular function following i.my. transplantation of PCMO as well as early (24 h post-infarction) i.v. application while nonmodulated ***monocytes*** failed to restore heart function. The Y-chromosome-specific SRY gene of male donor PCMO was detected exclusively in infarcted hearts of animals, which demonstrated improved cardiac function. Subdivision of infarcted hearts by microdissection localized the SRY gene-containing department to the left ventricle adjacent to the infarcted area whereas the right ventricle remained negative. Successful generation of PCMO in access numbers allows their autologous use as a new additive treatment for early restoration of cardiac function after MI.